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The Pulmonary Venous Systolic Flow Pulse—Its Origin and Relationship to Left Atrial Pressure

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OBJECTIVES The purpose of this study was to determine the origin of the pulmonary venous systolic flow pulse using wave-intensity analysis to separate forward- and backward-going waves.

BACKGROUND The mechanism of the pulmonary venous systolic flow pulse is unclear and could be a "suction effect" due to a fall in atrial pressure (backward-going wave) or a "pushing effect" due to forward-propagation of right ventricular (RV) pressure (forward-going wave).

METHODS In eight patients during coronary surgery, pulmonary venous flow (flow probe), velocity (microsensor) and pressure (micromanometer) were recorded. We calculated wave intensity ($dP \times dU$) as change in pulmonary venous pressure (dP) times change in velocity (dU) at 5 ms intervals. When $dP \times dU > 0$ there is a net forward-going wave and when $dP \times dU < 0$ there is a net backward-going wave.

RESULTS Systolic pulmonary venous flow was biphasic. When flow accelerated in early systole (S1), pulmonary venous pressure was falling, and, therefore, $dP \times dU$ was negative, -0.6 ± 0.2 ($x \pm SE$) W/m^2, indicating a net backward-going wave. When flow accelerated in late systole (S2), pressure was rising, and, therefore, $dP \times dU$ was positive, 0.3 ± 0.1 W/m^2, indicating a net forward-going wave.

CONCLUSIONS Pulmonary venous flow acceleration in S1 was attributed to a net backward-going wave secondary to a fall in atrial pressure. However, flow acceleration in S2 was attributed to a net forward-going wave, consistent with propagation of the RV systolic pressure pulse across the lungs. Pulmonary vein systolic flow pattern, therefore, appears to be determined by right- as well as left-sided cardiac events. (J Am Coll Cardiol 1999;34:802–9) © 1999 by the American College of Cardiology

Flow velocities in the extraparenchymal pulmonary veins typically demonstrate two major antegrade flow pulses, one during ventricular systole (VS) (S wave) and one during diastole (D wave). Clinical studies that have recorded pulmonary vein (PV) velocities by Doppler echocardiography suggest that the ratio between systolic and diastolic flow velocities may serve as a noninvasive estimate of left ventricular (LV) filling pressure (1–3). Thus, Kuecherer et al. (1) demonstrated that the systolic fraction of the pulmonary vein velocity-time integral was reduced in patients with elevated LV filling pressure and that an acute elevation of left atrial (LA) pressure was associated with a decrease in the systolic fraction. Similarly, it has been found that patients with elevated LV filling pressure have reduced peak systolic velocity and reduced ratio between peak S and D velocities (2–4). In direct contrast to this, studies in dogs have shown an increase in the S wave and the S/D ratio when LA pressure is increased by intravenous volume loading (5–7). These observations are difficult to reconcile but may reflect the dependency of the S wave on factors other than LA pressure.

The early diastolic PV flow wave is caused by LV relaxation and the subsequent opening of the mitral valve, and the velocity is determined largely by the factors that determine peak early transmitral filling (8). The origin of the PV S-wave, however, is less clear, and there is considerable disagreement in the literature. Several experimental studies in dog models conclude that the S-wave is generated predominantly by transmission of the right ventricular (RV) pressure pulse through the pulmonary vascular bed (9–12). Other studies done with dog models conclude that the systolic flow pulse in the PV is caused by pressure changes in the LA having a "suction" effect (13–16). This has been attributed to the effects of atrial relaxation and systolic descent of the atrioventricular (AV) plane, which decrease atrial pressure (13).

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Abbreviations and Acronyms

AS	= atrial systole
AV	= atrioventricular
A-wave	= pressure wave generated by the atrial systole
CABG	= coronary artery bypass grafting
CAD	= coronary artery disease
D	= diastole
dP × dU	= product of increments in pressure (dP) and velocity (dU) over 5 ms intervals
ECG	= electrocardiogram
LA	= left atrial or atrium
LV	= left ventricular or ventricle
Peak D flow rate	= peak pulmonary venous diastolic flow rate
Peak S flow rate	= peak pulmonary venous systolic flow rate
PV	= pulmonary vein or venous
RV	= right ventricular or ventricle
S	= systole
S1	= early systolic flow pulse
S2	= late systolic flow pulse
VS	= ventricular systole

The available data from humans do not establish which mechanism is causing the S-wave. This understanding is essential, however, for interpretation of PV flow data in a clinical context. Therefore, the main objective of this study was to determine the origin of the PV systolic flow pulse in patients with coronary artery disease (CAD) at baseline and during acute elevation of LV filling pressure. Pulmonary venous flow recorded by flowmeter and velocity recorded by intravascular sensor could be precisely timed to PV pressure recorded by micromanometer. To determine the origin of the flow pulses as being predominantly due to upstream (pulmonary vasculature and RV) and downstream (LA and LV) events, we performed wave-intensity analysis (17–19).

METHODS

Patients. The study population included eight patients who underwent elective coronary artery bypass surgery (CABG). There were seven men and one woman, with a mean age of 61 ± 12 (±SD) years. The patients had either double or triple vessel CAD. Mean LV ejection fraction was 52 ± 12 (±SD), ranging from 35% to 68%. Patients with unstable angina and patients with hemodynamic instability were excluded. The study was approved by the ethics committees at St. Paul's Hospital and the University of British Columbia. Informed written consent was obtained from each patient.

Preoperative medication included beta-adrenergic blocking agents in five patients, nitrates in five patients, calcium channel blocking agents in four patients and angiotensin-converting enzyme inhibitors in three patients. Two patients had diabetes mellitus and were on insulin.

Procedure. The patients were anesthetized using a balanced anesthetic technique, including sufentanil, midazolam, isoflurane and muscle relaxation. Ventilation was adjusted to maintain normal arterial blood gases and end-tidal (CO_2). The patients were ventilated with volume-controlled ventilators. The chest was opened by a median sternotomy.

The surgical procedure included sternotomy and wide pericardial split. The study was done just before initiation of extracorporeal circulation, after all preparations had been done, including insertion of an aortic cannula. An electrocardiogram (ECG) was recorded and was fed into the echocardiographic recorder and the computer system.

Recording of liquid-pressures. To serve as an absolute pressure reference for the micromanometers, a 16-gauge fluid-filled cannula was placed in the LA and was connected to an external pressure transducer. Mid-chest level was used as pressure zero. During zero adjustment, mean PV pressure was assumed to equal mean LA pressure. Because the PV pressure sensor was less than 2 cm into the vein, the error in this assumption was small.

Echocardiography. A Hewlett-Packard 5-MHz transesophageal echocardiographic probe (Hewlett-Packard Co., Palo Alto, California) was placed in the esophagus for measuring PV diameter.

Recording of PV flow. An 8- or 10-mm ultrasonic transit time flow probe (Transonics Systems, Ithaca, New York) was placed on the right lower PV close to its entrance into the LA. The probe was connected to a flowmeter (Transonics Systems, Ithaca, New York). Ultrasonic gel was applied to obtain good contact between the vein and the flowprobe.

Measurement of PV pressure and flow velocity. According to routine surgical procedure during cardiopulmonary bypass, the LV was vented via a PV. Therefore, the right lower PV was prepared with a small incision that could be used for introduction of the 7-F combined micromanometer and fluid velocity sensor. About 4.5 cm from the tip of the catheter, there was a micromanometer and an electromagnetic fluid velocity sensor (model SSD-827, Millar Instruments, Houston, Texas). The sensors were placed in the PV less than 2 cm from the entrance into the LA. For each patient the pressure sensor was calibrated before catheter sterilization and recalibrated at the end of each study. In each case the calibration factor (gain) was unchanged.

The velocity sensor was connected to a Carolina electromagnetic flowmeter (Model 501D, Carolina Medical Electronics, King, North Carolina). It was calibrated before each study using a constant flow system where saline was pumped into a plastic tube of 6-mm diameter. Flow rates were calculated from timed collections of saline in a graduated cylinder.

All ECG, pressure, velocity and flow data were digitized at 200 Hz and were stored on a computer for later analysis

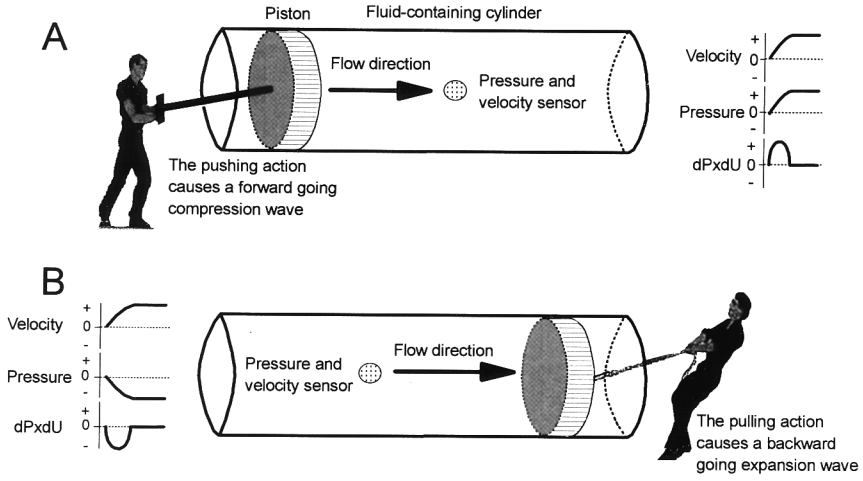

Figure 1. Schematic presentation of principles of wave intensity analysis: **(A)** Fluid inside a tube is set into motion by pushing an upstream piston by a constant force. The graph to the right displays velocity (U), pressure (P) and the product of increments in pressure (dP) and velocity (dU); i.e., dP × dU. Because there is a transient rise both in fluid pressure (+dP) and in velocity (+dU), dP × dU is positive. The +dP × dU indicates a forward-going wave and the +dP indicates a compression wave, thus a forward-going compression wave. **(B)** Fluid is set into motion by pulling a downstream piston. This causes a decrease in pressure (−dP), but an increase in velocity (+dU), resulting in a negative dP × dU. The −dP × dU indicates a backward-going wave, the −dP indicates an expansion wave, thus a backward-going expansion wave. The components of each of the four possible waves are outlined in Table 1.

using CVSOFT (Odessa Computers, Calgary, Canada). According to bench experiments, the time lag between the recorded pressure and ultrasonic transit time flow was 5 msec or less, and according to data from the producer of the combined micromanometer and electromagnetic fluid velocity sensor (Millar Instruments), the time lag was less than 2 ms.

Protocol. The first set of recordings was taken during baseline conditions. To study the effect of elevation of LA pressure, we infused saline via the aortic cannula, aiming at a peak mean LA pressure of about 18 mm Hg. An average of 700 ml was infused.

Calculations. In general the PV flowmeter gave higher quality signals than the electromagnetic velocity catheter. Adequate electromagnetic velocity signals were obtained in five patients. For the wave propagation analysis, we calculated velocity from the flow data by dividing by PV cross-sectional area. The area was calculated by measuring vein diameter by transesophageal echocardiography.

We calculated PV systolic flow fraction by dividing the systolic flow-time integral with the sum of the systolic and early diastolic flow-time integrals. The systolic flow-time integral was calculated from the onset of forward flow after the R-wave on the ECG to the nadir between the systolic and diastolic flow waves. The diastolic flow-time integral was calculated from the nadir between the systolic and diastolic flow waves to the start of the atrial wave (A-wave).

All measurements were done at end-expiration, and in each case, three consecutive beats were analyzed and averaged.

Wave intensity analysis. Wave intensity analysis has been described by Parker et al. (17,18) as a method of studying intravascular flow propagation. Wave intensity is calculated as the product of a change in pressure (dP) and change in velocity (dU) and was originally used to study wave propagation in the aorta. Wave intensity is a measure of the power transported by waves in a flow tube, although it is only a fraction of the total power of the working ventricle (17,18). The area under the dP × dU versus the time curve represents the energy transfer. The dP × dU has units of energy per square meter per second (joules/m^2/s, i.e., W/m^2).

The wave intensity at a given site in a given vessel contains information about both upstream and downstream events. By measuring simultaneous pressure and velocity in the vessel, it is possible to tell whether the net wave originates upstream or downstream from the point of interest.

Figure 1 is a simplified schematic presentation that illustrates some of the principles of wave intensity analysis: the upper panel (A) illustrates a forward-propagating wave inside a fluid containing cylinder, and the lower panel (B) illustrates a backward-going wave. See figure legends for explanation of the principles involved.

In these examples, positive direction is from left to right: upstream to downstream. Therefore, if the piston had been pushed left, a "backward-going compression wave" (not illustrated) would be generated. Table 1 summarizes interpretations made from the dP × dU analysis. It is important to be aware that the present analysis only tells the origin of the dominating or net wave.

Statistics. Data are reported as mean ± SEM. Values before and after volume loading were compared using a two-tailed paired Student *t* test. Regression analyses were performed using a least squares method and, when appro-

Table 1. Wave Characterization

Characterization	Force	Location of Force	dP	dU	dP × dU
Forward-going compression	Pushing	Upstream	+	+	+
Backward-going compression	Pushing	Downstream	+	−	−
Forward-going expansion	Pulling	Upstream	−	−	+
Backward-going expansion	Pulling	Downstream	−	+	−

dP = change in pressure; dU = change in velocity.

priate according to Glantz and Slinker (20), with a multiple-regression model including dummy variables to account for between-individual differences. A p value of less than 0.05 was considered significant.

RESULTS

Figure 2 displays PV pressure and velocity by flowmeter and by electromagnetic sensor. The PV flow velocity trace has two positive peaks during VS and one during ventricular D. During AS there is reversal of flow. Figure 3 shows a representative recording of PV pressure, flow velocity and dP × dU. Note that early systolic flow pulse (S1) starts when PV pressure starts to decline, (i.e., corresponding to atrial relaxation). Note also that onset of flow acceleration during the late systolic flow pulse (S2) is associated with a rise in PV pressure. Therefore, S1 and S2 acceleration are associated with directionally opposite changes in PV pressure. Biphasic systolic flow was seen in five of the patients during baseline, but S1 and S2 tended to merge after volume loading.

As demonstrated in Figure 3, there are several peaks in the dP × dU trace. During AS there is a rise in PV pressure and a fall in velocity, and dP × dU, therefore, is negative. This represents a backward-going compression wave. During early VS and corresponding to S1, there is a decrease in pressure and an increase in velocity, and dP × dU is negative. This represents a net backward-going expansion wave and is compatible with suction.

During late systole, however, when velocity continues to rise (S2), there is also a rise in pressure. This results in a positive dP × dU, indicating a net forward-going compression wave. When approaching peak V-wave pressure, there is another negative dP × dU, consistent with another backward-going compression wave.

During early diastole, PV pressure falls and velocity increases again (D wave) and the dP × dU becomes negative, indicating a net backward-going expansion wave. This corresponds in time to peak early transmitral filling.

Figure 4 shows wave intensity data from a patient during baseline and after volume loading. Volume loading mark-

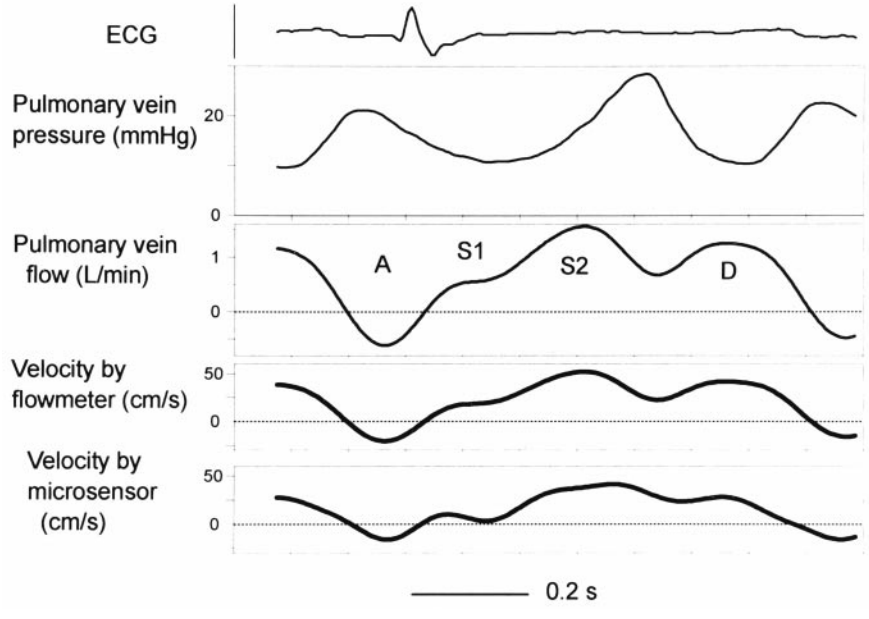

Figure 2. Representative traces showing pulmonary venous pressure and flow velocities by ultrasound transit time and by intravascular electromagnetic velocity sensor. Qualitatively similar velocity traces were obtained by either method. The figure shows the four pulmonary venous flow pulses, the A-wave during atrial contraction, the S1 and S2 waves during ventricular systole, and the D-wave early diastole. A = flow purse during atrial systole; D (diastole) = early diastolic flow pulse; S1 = early systolic flow pulse; S2 = late systolic flow pulse.

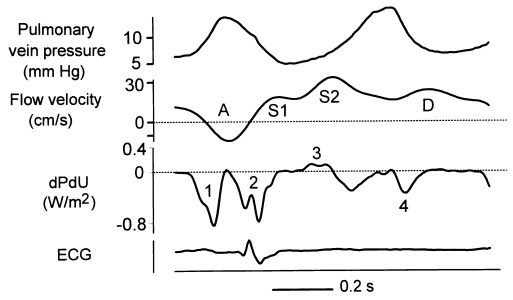

Figure 3. The figure shows the four pulmonary venous flow pulses, the A-wave during atrial contraction, the S1 and S2 waves during ventricular systole and the D-wave during early diastole. The numbers indicate four major waves in the dPdU trace. During atrial contraction the rise in pressure and decrease in velocity cause a negative dPdU **(1)**, indicating a net backward-going compression wave. During atrial relaxation and during early ventricular systole corresponding to S1, PV pressure is falling and velocity is rising. Therefore dPdU is negative **(2)**, indicating a backward-going expansion wave. Later in systole, corresponding to S2, PV pressure is rising and velocity is rising, and therefore dPdU is positive **(3)**. This indicates a net forward-going compression wave. During early diastole, PV pressure is falling and velocity is rising and dPdU is negative **(4)**, indicating a net backward-going expansion wave. dPdU = product of increments in pressure (dP) and velocity (dU) over 5 ms intervals; PV = pulmonary vein. Other abbreviations as in Figure 2.

edly increased the magnitude of all the dP × dU components, but there was considerable individual variability in the magnitude of the change (Table 2).

Ideally, the wave-intensity analysis should be done with directly measured velocities rather than velocities calculated from flow data. The velocity traces from the intravascular sensor, however, were often noisy, probably representing motion artifacts. In four patients with good quality dP × dU recordings from the intravascular velocity sensor, we did a comparison of the wave intensity pattern derived from the flowmeter recordings and from the intravascular sensor. The results were qualitatively similar using the two approaches. Thus, the dP × dU during S2 was positive in every patient, and with volume loading it increased from 0.2 ± 0.1 to 0.8 ± 0.1 W/m^2 ($p < 0.05$), calculated from the intravascular velocity data and from 0.3 ± 0.03 to 0.7 ± 0.07 W/m^2 ($p < 0.05$) when calculated from the flowmeter data. Similarly, during AS dP × dU was negative in every patient and decreased with volume loading from −0.4 ± 0.1 to −1.1 ± 0.3 W/m^2 ($p < 0.05$) and from −0.5 ± 0.08 to −1.7 ± 0.4 W/m^2 ($p < 0.05$), respectively.

Volume loading increased peak pulmonary venous systolic flow rate (peak S flow rate) as well as peak pulmonary venous diastolic flow rate (peak D flow rate) velocity and augmented flow reversal during atrial contraction (Fig. 4). The peak S/D ratio decreased with volume loading (Table 2). Similarly the ratio between the systolic (S1 + S2) and the combined systolic and early diastolic (D) flow integrals decreased from 0.70 ± 0.03 to 0.56 ± 0.06 ($p < 0.05$). For the pooled data, there were no significant correlations between the S/D ratio or the systolic flow fraction and mean LA pressure. When performing multiple regression in a model that included dummy variables to account for between-individual differences, the relationship between S/D ratio and LA mean pressure was statistically significant ($p < 0.001$). Similar results were obtained when this correlation was done between the systolic flow fraction and LA mean pressure ($p < 0.001$).

To determine whether the reduction in the S/D ratio was related to a change in LA chamber compliance, we calculated an index of LA compliance by integrating PV flow during LV systole, starting at the LA pressure nadir and ending at peak V-wave pressure. The operative compliance

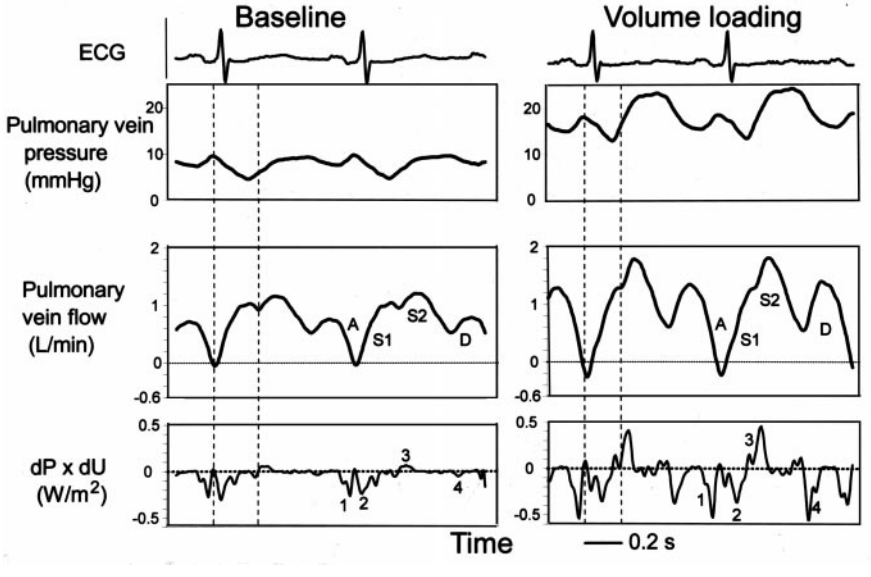

Figure 4. The effect of volume loading on pulmonary vein flow pattern and dPdU. Abbreviations as in Figures 2 and 3.

Table 2. Hemodynamic Variables at Baseline and at Elevated Preload

	Baseline	Volume Load	p Value
Mean flow rate (L/min)	1.10 ± 0.11	1.3 ± 0.33	
Peak S flow rate (L/min)	1.98 ± 0.22	2.64 ± 0.49	*
Peak D flow rate (L/min)	1.56 ± 0.24	2.54 ± 0.52	*
Minimum A flow rate (L/min)	0.06 ± 0.14	−0.77 ± 0.24	*
Ratio S/D flow rate	1.30 ± 0.09	1.07 ± 0.04	*
Systolic flow integral (%)	70 ± 3	56 ± 4	*
Mean flow rate (L/min)	1.10 ± 0.11	1.30 ± 0.29	*
PV mean pressure (mm Hg)	10.3 ± 1.9	18.6 ± 1.2	*
Heart rate (beats/min)	69 ± 6	71 ± 6	
Peak dP × dU during A (W/m^2)	−0.4 ± 0.3	−1.5 ± 0.4	*
Peak dP × dU during S1 (W/m^2)	−0.6 ± 0.2	−2.1 ± 0.6	*
Peak dP × dU during S2 (W/m^2)	0.3 ± 0.1	0.5 ± 0.1	*
Peak dP × dU during D (W/m^2)	−0.7 ± 0.3	−5.1 ± 2.0	*

*$p < 0.05$.

A = atrial; D = diastole; dP × dU = product of increments in pressure (dP) and velocity (dU) over 5 ms intervals; Minimum A flow rate = minimum pulmonary venous flow rate during atrial systole; Peak D flow rate = peak pulmonary venous diastolic flow rate; Peak S flow rate = peak pulmonary venous systolic flow rate; PV = pulmonary venous; S1 = early systolic flow pulse; S2 = late systolic flow pulse.

index calculated as change in volume divided by change in pressure decreased from 1.08 ± 0.27 to 0.47 ± 0.11 ml/mm Hg.

DISCUSSION

Previous studies on the contribution from left- and right-sided cardiac events to the systolic PV flow pulse are contradictory (7,9–16,21,22). The studies favoring a dominance from right-sided or upstream events (i.e., forward-going wave) have shown a close temporal relationship between the S wave and the flow wave in the pulmonary artery and capillaries (9–11). Furthermore, Appleton et al. (7) demonstrated that in conscious dogs the contour of the pressure trace in small PVs resembles that in the pulmonary artery. The studies that favor a dominance from pressure changes in the LA (i.e., backward-going wave) have based their conclusion on the observation that oscillations in PV flow follow a profile that is approximately the inverse of LA pressure and by showing that oscillations in PV flow are maintained when the influence from the right side is eliminated (13,15). These studies, however, cannot exclude contributions from forward-going waves.

Origin of the PV systolic flow pulses. To overcome the problem of distinguishing between forward- and backward-going waves, we applied wave intensity analysis (17–19). The qualitative result of this analysis can be predicted intuitively by considering pressure and velocity measured in the same location inside the vein. When velocity is increased by forward propagation of a positive pressure pulse, a simultaneous rise in PV pressure and velocity will be measured. This is analogous to the blood being "pushed" forward. In the case of velocity being increased by a suction effect or backward propagation of a negative pressure wave, a rise in velocity will be accompanied by a fall in pressure, analogous to the blood being "pulled" into the atrium. The latter effect was observed during atrial relaxation and during early ventricular systole. This backward going expansion wave was of substantial magnitude as indicated by the dP × dU.

Later in systole, however, when pulmonary flow velocity accelerated toward its peak value, PV pressure was rising, and dP × dU was positive. Therefore the second systolic flow acceleration cannot be attributed to a net suction effect. The combination of a rise in pressure and a rise in velocity must be attributed to a forward-going positive pressure pulse, compatible with transpulmonary propagation of the RV pressure pulse. It is important to note that this analysis only tells which mechanism is dominant. Therefore, it remains possible and is likely that a backward-going expansion wave (atrial suction) of lesser magnitude has contributed. It might be possible to estimate the contributions from forward- and backward-going waves (18), but this requires assumptions about wave speed. We did not measure wave speed and these calculations were not done.

In support of a "suction" effect, Smallhorn et al. (23) demonstrated a systolic PV flow pulse in patients who had undergone the Fontan procedure or after a Glenn shunt, which bypasses the RV. These patients, who have no pulmonary artery pulsations, did have a systolic PV flow pulse. In some patients LA pressure was recorded along with PV flow velocity. From their traces it is clear that systolic flow acceleration occurs when atrial pressure is falling (i.e., during atrial relaxation consistent with a suction mechanism). Therefore, the findings in Fontan patients are entirely consistent with the interpretations of the present data (i.e., that a suction effect from the LA contributes to the early systolic flow pulse). However, the conclusions by Smallhorn et al. (23) that transmission from the RV plays no role in flow in the extraparenchymal PVs is not sup-

ported by our data. Their observation that a systolic flow component was present after a Fontan procedure or a Glenn shunt implies that left-sided cardiac events contribute, but do not rule out, a contribution from right-sided events in patients with normal vascular connections.

This study, therefore, demonstrates that the systolic PV flow pulse has an early phase that can be attributed to a suction effect created by atrial relaxation and possibly also by descent of the AV plane during early systole. The second phase, during which velocity reaches its peak, however, is attributed to a net forward-going compression wave that most likely is generated by the RV. A "suction" effect from the LA may have contributed, but the dominant mechanism is the forward "pushing" effect. For the remaining part of the cardiac cycle, the PV flow pulsations are attributed almost entirely to downstream events, i.e., the marked backward-going expansion wave during early VS and during mitral opening and the backward-going compression waves during atrial contraction and during late VS. Following intravenous volume loading, all of the components in the wave intensity analysis increased in magnitude.

The present study did not consider the possibility that reflected waves may have contributed to the PV flow pattern. It is possible that pressure waves that originate in the LA and travel backward toward the pulmonary vascular bed are reflected and then propagate forward toward the atrium. In particular, the pressure wave generated by the atrial systole (A-wave) may travel toward the pulmonary vascular bed, then the reflected wave becomes part of the S-wave. In the studies of Skagseth (24) of patients with atrial fibrillation, however, there was still a late systolic PV flow pulse. Therefore, the reflected A-wave may have contributed to, but cannot be the only mechanism of, the late systolic forward-going wave.

Relationship between PV flow pattern and mean LA pressure. We confirmed the observations of Kuecherer et al. and others (1–3) that the ratio between PV S and D wave decreased with volume loading. It has been suggested that this response is related to reduced operating compliance in the LA when intracavitary pressure is elevated (1). This relationship was demonstrated in this study using the PV flow integral to estimate changes in atrial compliance. Because this method includes only one of the four veins, it does not give a true atrial pressure-volume relationship, but reflects the apparent compliance relevant to the vein under investigation. Because none of the patients had more than mild mitral regurgitation, this was probably not a major limitation. The wave-intensity analysis showed a marked backward-going compression wave during late systole. This is interpreted as a reflected wave caused by the high systolic PV flow rate that results in a marked pressure rise in the atrium. In Figure 3, this $dP \times dU$ component is seen after the peak of S2.

The systolic PV flow pulse is created by the combined effect of the pressure fall in the LA (which is a function of LA and possibly LV properties) and the pressure and flow wave generated by the RV and possibly in part by reflected waves. In addition, resistance and capacitance of the pulmonary vasculature will probably be of importance. Therefore, it is not surprising that the effect of elevated preload on the S/D ratio varies in different models and clinical settings. Of particular interest is the experimental study by Hoit et al. (5) that showed that elevation of LA pressure by volume loading was associated with increased S/D ratio, while elevation of LA pressure by cardiodepression gave the opposite effect. The shift in the relationship with cardiodepression could reflect reduced RV stroke volume as well as reduced LA and LV systolic function.

There does not seem to be a strong relationship between LA mean pressure and the S wave, but rather between RV, LA and LV systolic function and the S wave. Depression of RV function will reduce the intensity of any forward-propagating pulse, and, therefore, may reduce the S2 wave. A reduction in LA systolic function will tend to reduce the S1 wave, due to smaller atrial pressure wave and, therefore, less marked atrial pressure fall during early VS. Reduction of LV function will reduce the S wave due to less marked systolic descent of the AV plane (21,23). A mechanism by which elevated LA mean pressure will tend to reduce the S wave is by reducing atrial chamber compliance. As suggested by the calculated index of LA compliance, this mechanism may have contributed.

Study limitations. The patients were studied while under anesthesia with an open chest. This may have influenced the flow pattern. However, because the flow traces were similar to those recorded in patients who were awake, we believe that the conclusions in this study are likely to be valid for those patients as well. It is possible that the degree of transpulmonary wave propagation is different with the chest open or closed, but the principles should be the same.

The patient population in this study was small and somewhat inhomogeneous. This limited our ability to give reliable estimates of the absolute magnitude of the various components in the $dP \times dU$ trace. It is also possible that the relative magnitude of the different components will vary with the functional status of the heart.

Conclusions. In conclusion, this study demonstrated that S1 is associated with a fall in PV pressure and is compatible with a suction effect from the LA and LV, probably due to the combined effect of atrial relaxation and descent of the AV ring during early systole. The S2, however, is associated with a rise in PV pressure and, therefore, cannot be attributed to a net suction mechanism. It is most likely caused in part by forward propagation of the RV systolic pressure pulse. Further studies should be done to determine the potential contribution from reflected waves.

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